**TCT-268**

**Incidence and predictors of tissue prolapse after percutaneous coronary intervention for saphenous vein graft disease: Intravascular ultrasound study**

*Young Joon Hong[1], Myung Ho Jeong[1], Yun Ha Choi[1], Soo Young Park[1], Hae Chang Jeong[1], Jae Yeong Cho[1], Sang Cheol Cho[1], Jong Hyun Yoo[1], Su Young Jang[1], Ji Eun Song[1], Doo Sun Sim[1], Keun Ho Park[1], Ju Han Kim[1], Youngkeun Ahn[1], Jeong Gwan Cho[1], Jong Chun Park[1], Jung Chaee Kang[1]*
*[1]Heart Research Center, Chonnam National University Hospital, Gwangju, Korea, Republic of*

**Background:** The aim of this study was to investigate the relationship between intravascular ultrasound (IVUS) findings and the no-reflow phenomenon and long-term outcome after percutaneous coronary intervention (PCI) of saphenous vein graft (SVG) lesions.
**Methods:** No-reflow was defined as post-PCI TIMI grade 0, 1, or 2 flow.
**Results:** Of 311 patients who underwent pre- and post-stenting IVUS, no-reflow was observed in 39 patients (13%). Degenerated SVG (62% vs. 36%, p=0.002) was observed more frequently in the no-reflow group. IVUS-detected intraluminal mass (82% vs. 43%, p<0.001), culprit lesion multiple plaque ruptures (23% vs. 6%, p<0.001), and tissue prolapse (51% vs. 35%, p=0.043) were significantly more common in patients with no-reflow. In the multivariate logistic regression analysis, an intraluminal mass (Odds ratio [OR]=4.84; 95% CI 1.98-10.49, p=0.001), culprit lesion multiple plaque ruptures (OR=3.46; 95% CI 1.46-8.41, p=0.014), and degenerated SVGs (OR=3.17; 95% CI 1.17-6.56, p=0.024) were the independent predictors of post-PCI no-reflow. At 5-year clinical follow-up, the rates of death [14 (36%) vs. 55 (20%), p=0.036] and myocardial infarction [13 (33%) vs. 52 (19%), p=0.039] were significantly higher in the no-reflow group. However, the rate of target vessel revascularization was not different significantly between two groups [15 (38%) vs. 90 (33%), p=0.3].
**Conclusions:** IVUS-detected intraluminal mass, multiple plaque ruptures, and degenerated SVGs are associated with post-PCI no-reflow in SVG lesions. No-reflow was associated with poor long-term clinical outcomes after PCI for SVG lesions.

**TCT-269**

## Abstract Withdrawn

**TCT-270**

## Abstract Withdrawn

**TCT-271**

**Stent Thrombosis After Intravascular Ultrasound-guided Stent Implantation**

*Yoshihide Fujimoto[1], Masashi Yamamoto[2], Yoshio Kobayashi[1]*
*[1]Chiba University Graduate School of Medicine, Chiba, Japan, [2]Kimitsu Central Hospital, Kisarazu, Japan*

**Background:** Intravascular ultrasound (IVUS) is used frequently for percutaneous coronary intervention (PCI) in Japan. However, there is little information about the incidence of stent thrombosis after IVUS-guided stent implantation.
**Methods:** Between January 2005 and December 2011, 2,992 lesions in 2,685 patients underwent IVUS-guided bare metal stent (BMS) (n=981) and drug-eluting stent (DES) implantation (n=2,011). The Academic Research Consortium definition of stent thrombosis was used.
**Results:** Definite stent thrombosis was observed in 10 lesions (1.0%) with BMS (early 0.9%, late 0.1%, and very late 0%) and 9 lesions (0.4%) with DES (early 0.2%, late 0%, and very late 0.2%). Stent thrombosis occurred in 12 (1.6%) and 7 lesions (0.4%) that had undergone PCI for acute myocardial infarction and other indications, respectively. Stent underexpansion was observed in 6 (46%) of the 13 lesions with early stent thrombosis. On the other hand, none of the 6 lesions with late or very late stent thrombosis had stent underexpansion.
**Conclusions:** IVUS-guided stent implantation results in a low incidence of stent thrombosis. Stent underexpansion is a risk factor of early stent thrombosis, but it was not for late or very late stent thrombosis.

**TCT-272**

**The Role of Macrophage Accumulations on Bare-Metal Stent Failure Presenting With Acute Coronary Syndrome: Observation by Optical Coherence Tomography**

*Yuetsu Kikuta[1], Hideo Takebayashi[1], Shigeki Hiramatsu[1], Manabu Tanigichi[1], Kenji Goto[1], Masahito Taniguchi[1], Katsumasa Sato[1], Etsuko Ikeda[1], Arata Hagikura[1], Hiroki Yamane[1], Seiichi Haruta[1]*
*[1]Fukuyama Cardiovascular Hospital, Fukuyama, Hiroshima*

**Background:** Little is known about the role of macrophage accumulations (Mφ) on bare-metal stent (BMS) failure.
**Methods:** We evaluated 43 consecutive BMS failure lesions in 43 patients (11 acute coronary syndrome [ACS] and 32 stable myocardial ischemia [SMI], median stent duration 9.6 [interquartile range 6.9-58.8] months) by optical coherence tomography. Neointima with Mφ was defined as thin bright layer with shadowing (peak intensity >180 and attenuation rate >2, when measurements were fitted to an approximate exponential function).
**Results:** The mean age was 67.4±10.0 years, 40 patients (93.0%) were male, and 16 (37.2%) were diabetic. Compared with SMI patients, ACS patients showed higher incidence of thin-cap fibroatheroma (TCFA)-containing neointima (63.6% versus 6.3%, p=0.0003), lesions with Mφ (72.7% versus 9.4%, p=0.0001), neointimal rupture (72.7% versus 15.6%, p=0.001), thrombus (81.8% versus 43.8%, p=0.039), and had higher admission LDL (116.4±26.6 versus 90.8±23.9 mg/dl, p=0.012) and lower HDL cholesterol level (39 [range 33-45] versus 47 [40-55] mg/dl, p=0.021). Stent failure with TCFA-containing neointima (n=9) expressed higher incidence of Mφ (88.9% versus 8.8%, p<0.0001), larger Mφ angle (143 [range 84-262] versus 0 [0-0] °, p=0.002), and longer Mφ longitudinal length (8 [range 5-13] versus 0 [0-0] mm, p=0.003) than non–TCFA-containing neointima (n=34). Fibrous cap thickness negatively correlated with Mφ longitudinal length (r=−0.00449, p=0.045). Compared with lesions without neointimal rupture (n=30), lesions with neointimal rupture (n=13) demonstrated higher incidence of Mφ (69.2% versus 6.7%, p<0.0001). Thrombotic stent failure lesions (n=23) showed more Mφ (39.1% versus 10.0%, p=0.039) than non-thrombotic lesions (n=20). Eleven stent failure patients with Mφ presented later than 32 patients without Mφ: 104.8 (range 58.8-142.4) versus 9.0 (6.6-13.3) months (p=0.0004). Using receiver-operating curve analysis, 16.7 months was the best predictor of the presence of Mφ with a sensitivity of 100% and a specificity of 84% (area under curve =0.960, p<0.0001).
**Conclusions:** Mφ might be associated with neoatherosclerosis and unstable features of BMS neointima.

**TCT-273**

**Serial Evaluation Of Peri-strut Low Intensity Area On Optical Coherence Tomography After Drug-eluting Stents Implantation**

*Eui Im[1], Byeong-Keuk Kim[2], Jung-Sun Kim[3], Dong-Ho Shin[4], Young-Guk Ko[5], Donghoon Choi[6], Yangsoo Jang[2], Myeong-Ki Hong[7]*
*[1]Severance Cardiovascular Hospital, Seoul, Korea, Republic of, [2]Yonsei university, Seoul, Korea, Republic of, [3]Yonsei University, Seoul, Korea, Republic of, [4]Severance Cardiovascular Hospital, N/A, [5]Younsei University, Seoul, Korea, Republic of, [6]Severance Hospital, Yonsei University, Seoul, Korea, Republic of, [7]Yonsei Cardiovascular Hospital, Seoul, South Korea*

**Background:** Recent studies have demonstrated that peri-strut low intensity area (PLIA) seen on optical coherence tomography (OCT) represents neointimal fibrinogen and/or extracellular matrix and is associated with neointimal thickening (NIT) after 1st-generation drug-eluting stents (DES) implantation. However, there are no data regarding PLIA in new generation DES and its change in serial OCT evaluations.
**Methods:** A total of 83 patients underwent 9-month OCT after DES implantation (25 sirolimus-eluting stents [SES], 20 paclitaxel-eluting stents [PES], 30 zotarolimus-eluting stents [ZES], and 8 everolimus-eluting stents [ZES]). PLIA on OCT was defined as a region around stent struts with homogenous lower intensity than surrounding tissue without signal attenuation. Inter-stent analysis was performed between PLIA + and PLIA – stents, and then intra-stent analysis was performed between PLIA + and overall stent segments. The patients also underwent 2-year OCT evaluations and serial change of NIT was analyzed.
**Results:** The incidence of PLIA + stents on 9-month OCT was highest in PES and lowest in SES (90% in PES vs. 28% in SES vs. 60% in ZES vs. 63% in EES, p <0.001). In inter-stent analysis, PLIA + stents showed higher mean NIT than PLIA – stents (20.11 ± 9.69 μm vs. 10.86 ± 5.46 μm, p <0.001). Also in intra-stent analysis, PLIA + segments showed higher mean NIT than overall stent segments (25.79 ± 12.22 μm vs. 20.11 ± 9.69 μm, p = 0.013). On serial 2-year OCT, PLIA + stents showed smaller increase of NIT than PLIA – stents in inter-stent analysis (3.46 ± 10.50 μm vs. 7.41 ± 10.07 μm, p = 0.092). Also in intra-stent analysis, PLIA + segments showed smaller increase of NIT than overall stent segments (1.76 ± 11.63 μm vs. 3.46 ± 10.50 μm, p = 0.451). All of these findings were regardless of DES type.
**Conclusions:** Although PLIA was associated with increased NIT regardless of DES type, it may mean weak potential for further neointimal growth in long-term follow-up.

**TCT-274**

**Relationship Between Arterial Remodeling, Fibrous Cap Thinning and Lipid Accumulation: A Serial Integrated Backscatter Intravascular Ultrasound and Optical Coherence Tomography Study**

*Teruyoshi Kume[1], Hiroyuki Okura[1], Kenzo Fukuhara[1], Yoshinori Miyamoto[1], Shintaro Nezuo[1], Yoji Neishi[1], Akihiro Hayashida[1], Takahiro Kawamoto[1], Kiyoshi Yoshida[1]*
*[1]Kawasaki medical school, Kurashiki City, Japan*

**Background:** A serial intravascular ultrasound (IVUS) and optical coherence tomography (OCT) study has shown that positive arterial remodeling was related to thinning of fibrous cap. Serial changes in tissue components associated with fibrous cap and/or arterial remodeling is unknown Therefore, the purpose of this study was to evaluate the relationship between changes in fibrous cap thickness and changes in plaque tissue components by using optical coherence tomography (OCT) and integrated backscatter IVUS (IB-IVUS).
**Methods:** Serial (baseline and 6 months follow-up) IB-IVUS and OCT examinations were performed on 81 vessels from 56 patients with ischemic heart disease who underwent percutaneous coronary intervention. 81 fibroatheromas were selected from 48 culprit lesions and 33 non-culprit lesions. Serial changes and relationships between